

U.S. Department of Labor

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Issue Date: 28 July 2005

**CASE NO.: 2004-BLA-98
(Formerly 2001-BLA-01092
& 2001-BLA-01093)**

IN THE MATTER OF:

**FRANCES E. POOLE, Widow of
STERLING POOLE, Deceased Miner
Claimant**

v.

**FREEMAN UNITED COAL MINING CO.
Employer**

And

**DIRECTOR, OFFICE OF WORKERS'
COMPENSATION PROGRAMS
Party-in-Interest**

APPEARANCES:¹

Robert Henry Sarpy, ESQ.
For the Claimant

Shannon L. Clark, ESQ.
For the Employer

BEFORE: Lee J. Romero, Jr.
Administrative Law Judge

DECISION AND ORDER DENYING BENEFITS

This is a Decision and Order arising out of two claims for benefits under Title IV of the Federal Coal Mine Health and

¹ The Director, Office of Workers' Compensation Programs, was not present or represented at the hearing.

Safety Act of 1969, as amended by the Black Lung Benefits Act of 1977, 30 U.S.C. §§ 901-962, (hereinafter referred to as "the Act") and the regulations thereunder at Title 20 of the Code of Federal Regulations (CFR). Regulation section numbers mentioned in this Decision and Order refer to sections of that Title.

On July 27, 2001, this case was referred to the Office of Administrative Law Judges by the Director, Office of Workers' Compensation Programs, for a hearing. (DX-48).² A first formal hearing on this matter was conducted on January 14, 2002, in Metairie, Louisiana, by the undersigned Administrative Law Judge and a Decision and Order denying benefits issued on September 10, 2002.

On September 17, 2003, the Benefits Review Board issued a Decision and Order vacating the undersigned's original Decision and Order denying benefits and remanded the case to the District Director for further development of evidence consistent with its opinion. The Director, OWCP, requested a remand because of a failure to provide Claimant with a complete credible pulmonary examination addressing the existence of legal pneumoconiosis and whether Claimant's total disability is due to pneumoconiosis. (DX-64, p. 2). The District Director requested that Dr. Cecile Rose complete a medical opinion of Mr. Poole, upon which the District Director granted Claimant's requested modification on March 16, 2004. (DX-68). Subsequently, this matter was referred to the Office of Administrative Law Judges on April 14, 2004, and a second formal hearing was held on February 10, 2005, in Metairie, Louisiana.

On May 3, 2005, after filing post-hearing briefs regarding the second hearing in this case, Claimant filed a Motion and Order to Amend Claimant's Exhibit List to offer the curriculum vitae of Drs. Hebert and Winkler into the record.

On May 20, 2005, Employer filed an Opposition to Claimant's Motion to Amend Exhibits, noting the request to submit additional exhibits was untimely and would cause prejudice. More specifically, Employer contends it did not have the opportunity to review the curriculum vitae before filing its

² In this Decision, "CX-" refers to Claimant's exhibits, "DX-" refers to the Director's Exhibits, "EX-" refers to the Employer's Exhibits, "ALJX-" refers to the Administrative Law Judge's Exhibits, and "1Tr.; DX-56" refers to the official transcript of the first formal hearing and "2Tr." refers to the official transcript of the second proceeding.

post-hearing brief and, thus, did not have an opportunity to submit arguments with respect to the doctors' qualifications to render opinions in this matter. I find the curriculum vitae are necessary for a proper evaluation of the medical opinions of record. I further find that Employer would not be prejudiced by the acceptance of these documents into the record as the documents offer no additional factual information regarding the case and are relevant only with respect to the weight to be afforded the physicians' opinions. Accordingly, Claimant's Motion to Amend her exhibit list is granted and the curriculum vitae of Drs. Hebert and Winkler are entered respectively into the record as Claimant's exhibits Nos. 19 and 20.

All parties were afforded the opportunity to call and examine witnesses, to cross-examine witnesses and to present evidence, as provided in the Act and the above-referenced regulations. Post-hearing memoranda were received from Claimant and Employer.

ISSUES

The issues in this case are:

1. Whether the miner had pneumoconiosis as defined by the Act and regulations;
2. Whether the miner's pneumoconiosis arose out of coal mine employment;
3. Whether the miner's disability was due to pneumoconiosis; and
4. Whether the miner's death was due to pneumoconiosis.

(DX-46, 47; Tr. 21-24).

Miner/Claimant contend they have established the miner suffered from pneumoconiosis which arose out of coal mining employment and that the pneumoconiosis contributed to the miner's total disability and hastened his death. According to Miner/Claimant, the miner established the existence of "legal pneumoconiosis" through x-ray findings, a favorable autopsy, and the medical opinions of Dr. Rose, Dr. Hebert, and Dr. Winkler. Claimant contends that Employer's doctors focused strictly on a "clinical diagnosis" of pneumoconiosis.

Employer contends the evidence of record establishes that the miner did not suffer from coal workers' pneumoconiosis and that his disabling lung impairment was caused by his long-term smoking history.

Based upon a thorough analysis of the entire record in this case, with due consideration accorded to the arguments of the parties, applicable statutory provisions, regulations, and relevant case law, I hereby make the following:

FINDINGS OF FACT

Background:

The miner, Sterling Poole, filed an application for Black Lung benefits on January 20, 1999. (DX-1). On October 19, 1999, the District Director found Mr. Poole entitled to benefits. (DX-25). The employer controverted the award on October 29, 1999. (DX-26). On November 16, 1999, the claim was referred to the Office of Administrative Law Judges. (DX-28). Thereafter, Mr. Sterling died on November 15, 2000, and the claim was remanded on December 14, 2000 to allow Mrs. Poole to file a survivor's claim. (DX-30).

Mrs. Poole filed a survivor's claim on December 14, 2000. (DX-31). The District Director found Mrs. Poole entitled to benefits on June 1, 2001. (DX-43). The employer requested a hearing before the Office of Administrative Law Judges on June 25, 2001. (DX-44). On July 27, 2001, the claims were transferred to the Office of Administrative Law Judges. (DX-46).

Mr. Poole was born on September 25, 1929 and was seventy-one years old at the time of his death on November 15, 2000. (DX-1, DX-35). Mr. Poole completed three years of college. (DX-1). He married Frances Eloise Shaw on November 12, 1955, and she remained his only dependent. (DX-1, DX-33). Mrs. Poole was born September 20, 1937, and has no dependents. (DX-1, DX-34).

Mrs. Poole provided a three-page, handwritten letter describing her husband's condition prior to his death. (DX-54). Quoted below are its most salient points:

Sterling was a proud and independent person before he became ill. His independence was taken from him due to his illness.

In 1997 Sterling noticed a change in his health, from 1977 to 1999 and until his death on Nov. 15, 2000 the disease rapidly progressed. He had to depend on machines and medication to keep him alive.

He was unable to drive or go anywhere without assistance, due to his breathing problem and heart condition.

When Sterling retired at 62 years old it was not because of retirement age as stated in one report. It was due to health with severe coughing spells. At times he would black out and rather [than] be a danger to others or himself, he retired.

Sterling was hospitalized 3 times due to his illness. Two times he came home. His health never improved or the illness cured. All that could be done and provided was comfort measures.

The third hospitalization Sterling passed away.

It [has] been stated that due to my husband's smoking it was the contributing factor in his death. I disagree with it.

There is medical data stating smoking does not increase the prevalence of this disease, the area of the mine where the miner worked involving the dust is a contributing factor, and pneumoconiosis . . . [is a] chronic disease of the lung. "Black Lung" can take up to 25 years to develop after leaving the mines.

As the disease progresses the person develops [sic] loss of lung function and a heart deficiency called cor pulmonale which develop[s] in severe cases.

It is documented Sterling had all the signs and symptoms of Black Lung. His illness progressed rapidly to the point where he was on oxygen 24 hours a day, loss of weight and

appetite, had difficulty sleeping and was unable to walk any distance.

I read the reports from doctors Freeman Coal Co. hired. I am not saying they aren't qualified in their field. I do disagree with their reports on their findings on Sterling.

They never met Sterling, knew him as a person, or treated my husband for his illness. All they knew was medical reports and x-rays of Sterling.

Testimonial Evidence

Mrs. Poole testified at the first hearing that Mr. Poole began coal mining in 1949, before she even knew him. He worked for Old Ben Coal company. (1Tr. 27). After they were married, he worked for Inland Steel from 1970 to 1975. He then worked two years for Mrs. Poole's father in Pennsylvania, strip mining. Thereafter, Mr. Poole went to work for Freeman Coal Company for five years, ending in 1982. (1Tr. 27-28). He was a continuous mine operator, and the masks he wore were always dirty at the end of the day. (1Tr. 29). The employer did not contest Mr. Poole's assertion of fourteen years of coal mine employment, and I found that he was employed as a coal miner for fourteen years, lastly as a continuous miner operator. (1Tr. 46-47).

In 1997, the miner began to experience shortness of breath, feeling "down in the dumps," and developed a cough. (1Tr. 31). Mrs. Poole testified that her husband was smoking when she first met him in 1955 and continued to smoke even after he left coal mining. (1Tr. 31-32). However, according to Mrs. Poole, Mr. Poole was never a heavy smoker because he could not smoke in the mines, so during his coal mining years, he smoked only two or three cigarettes a day. (1Tr. 32). Mrs. Poole stated that her husband was hospitalized three times for his breathing problems, first in March 1999. (1Tr. 33). He was told that he only had 30% lung capacity remaining. (1Tr. 34). During the process of undergoing respiratory treatments, Mr. Poole suffered a heart attack in 1998 and was told it was related to his lungs. (1Tr. 34). He was treated with antibiotics, steroids, breathing treatments, and oxygen. (1Tr. 35). His family physician was Dr. Winkler. (1Tr. 36).

Tom Austin

Mr. Austin was deposed on August 20, 2004. (EX-53). He began working in Employer's safety department as an assistant safety director in December 1975, became the acting Safety Director, and then worked as a safety administrator. (EX-53, pp. 7-8). Mr. Austin made frequent visits to each of Employer's mines, during which he would "go down into the mine, view the operation, view the ventilation, do roof control studies, dust control work." (EX-53, p. 11).

Mr. Austin was familiar with the Mine Safety and Health Administration (MSHA) regulations related to mine safety and dust control, including the safety measures in place for the Orient 3 from 1977 to 1982. (EX-53, pp. 11-12). From 1977 to 1980, Employer "sampled" each person every other month to determine compliance with a two milligram dust standard. From 1980 to 1982, Employer performed "area sampling" of the "dust generation sources," namely the continuous miners and areas along the conveyor belt. Occasionally, Employer would sample roof boulders. (EX-53, pp. 12-13). Employer also performed its own testing, including "experimental samples" to determine if dust control measures were working properly. He testified that Orient 3 was predominantly in compliance with the two milligram standard between 1977 and 1982. (EX-53, p. 13).

In addition to sampling, Employer's dust control measures included sweeping fresh air over the miners at an average of 60 feet per minute. The air would be picked up by tubing attached to an Airdyne fan. During the process, water was sprayed "on the coal to control the dust and keep it up to the face where the fan could pick it up." Employer also offered two different respirators at no charge to anyone who requested it. (EX-53, p. 14).

Medical Evidence

A. Chest X-Rays:

X-ray Date	Exhibit No.	Physician	Reading
9-28-1982	EX-36 DX-52	Dr. Hutchinson	A few scattered calcified granuloma in both lungs; no evidence of pulmonary infiltrates or pleural effusions
8-25-1997	DX-42	Dr. Kerber	Severe chronic obstructive

			pulmonary disease; right upper lobe scarring; aortic atherosclerotic calcification
8-25-1997	DX-42	Dr. Main ³	Negative for pneumoconiosis; a fibrocalcific density in right apex consistent with granulomatous change; emphysema
8-25-1997	DX-42 DX-52	Dr. Wheeler ⁴	No evidence of silicosis or coal workers' pneumoconiosis; moderate emphysema with hyperinflation; minimal healed tuberculosis with calcified granulomata and focal fibrosis in posterior right apex and few tiny calcified granulomata in periphery left upper lobe and right lower lateral lung
10-2-1997	DX-42	Dr. Kerber	Old granulomatous disease; severe chronic obstructive pulmonary disease
10-2-1997	DX-42 DX-52	Dr. Main	Negative for pneumoconiosis; a fibrocalcific density in right apex consistent with granulomatous change; emphysema
10-2-1997	DX-42 DX-52	Dr. Wheeler	No evidence of silicosis or coal workers' pneumoconiosis; moderate emphysema with hyperinflation; minimal healed tuberculosis with calcified granulomata and focal fibrosis in posterior right apex and few tiny calcified granulomata in periphery left upper lobe and right lower lateral lung
6-15-1998	DX-42	Dr. Stephen Williams	Moderate to severe bilateral chronic obstructive pulmonary disease with scattered bilateral granuloma type

³ Dr. Main is a "B" reader and board-certified in radiology. A "B" reader is a physician who has demonstrated proficiency in assessing and classifying x-ray evidence of pneumoconiosis by successful completion of an examination conducted by or on behalf of the Department of Health and Human Services. Physicians' qualifications are a matter of public record at the HHS National Institute of Occupational Safety and Health (NIOSH) reviewing facility at Morgantown, West Virginia. (42 C.F.R. § 37.51). Consequently, greater weight is given to a diagnosis by a "B" Reader. See Blackburn v. Director, OWCP, 2 B.L.R. 1-153 (1979).

⁴ Dr. Wheeler is a "B" reader and Board-certified in radiology.

			nodules and scars; no definite acute or neoplastic chest disease or cardiomegaly
6-15-1998	EX-10 DX-52	Dr. Wheeler	No evidence of silicosis or CWP; moderate emphysema; minimal healed TB more likely than healed histoplasmosis with focal fibrosis and small calcified granuloma lower right apex
6-15-1998	EX-11 DX-52	Dr. Worrell ⁵	0/1; r/r; 4 zones; many of the round opacities are calcified and may represent granulomata versus pneumoconiosis
3-15-1999	DX-15	Dr. Preger ⁶	2/1; t/q; 3 zones; type A large opacities; bilateral healed granulomatous disease, question histoplasmosis; ill-defined large opacity in left lower lung; appearance is atypical for both CWP and asbestosis; history is important
3-15-1999	EX-12 DX-52	Dr. Wheeler	No evidence of silicosis or CWP; ill-defined infiltrate left lower lung near apex of heart compatible with edema or pneumonia or fibrosis; moderate emphysema; minimal healed TB with scars in right apex and few scattered calcified granulomata
3-15-1999	EX-13 DX-52	Dr. Worrell	1/1; q/p; 6 zones; many of these nodules are calcified and could represent granulomatous disease vs. pneumoconiosis; the coalescent pattern at the left base is non-specific, but pneumonia should be considered
3-16-1999	EX-14 DX-52	Dr. Wheeler	Ill-defined infiltrate or fibrosis left lower lung; emphysema; minimal healed TB with scar and calcified granuloma right apex and few calcified granulomata in both

⁵ Dr. Worrell is a "B" reader and Board-certified in radiology.

⁶ Dr. Preger is a "B" reader.

			lungs and probably in left hilum
3-16-1999	EX-15	Dr. Worrell	1/0; q/r; 2 zones; many of the opacities are calcified and may represent granulomata vs. pneumoconiosis
3-16-1999	CX-16	Dr. Stephen Williams	Probable acute mild left basilar pneumonia; COPD
3-20-1999	DX-42	Dr. Miller	Pneumonia; chronic obstructive pulmonary disease; no evidence of active/acute pulmonary disease
3-20-1999	EX-16 DX-52	Dr. Worrell	0/1; r/r; 4 zones; many of the round opacities are calcified and may represent granulomata vs. pneumoconiosis
3-20-1999	EX-17 DX-52	Dr. Wheeler	No evidence of silicosis or CWP; possible focal infiltrate in anterior inferior right middle lobe; moderate emphysema with hyperinflation; minimal healed TB with subtle right apical pleural thickening, focal fibrosis, and probable small calcified granuloma lower right apex and few tiny calcified granulomata
9-13-1999	EX-33 DX-52	Dr. Wester	Chronic obstructive pulmonary disease; scattered calcified granulomas; right apical density which may represent an area of parenchymal scarring, but a pulmonary nodule cannot be excluded
9-13-1999	EX-18 DX-52	Dr. Wheeler	No evidence of silicosis or coal workers' pneumoconiosis; moderate emphysema; minimal healed TB with coarse scar and probable small calcified granuloma
11-19-1999	DX-42	Dr. Matthews ⁷	0/1; s/t; 4 zones; emphysema; no acute cardiopulmonary disease
11-19-1999	EX-21 DX-52	Dr. Hippensteel ⁸	Negative for pneumoconiosis; a few scattered calcified granulomas; emphysema
11-19-1999	EX-22 DX-52	Dr. Castle ⁹	Negative for pneumoconiosis; granulomatous disease; emphysema
11-19-1999	EX-20	Dr. Wheeler	No evidence of silicosis or

⁷ Dr. Matthews is a "B" reader and Board-certified in radiology.

⁸ Dr. Hippensteel is a "B" reader.

⁹ Dr. Castle is a "B" reader.

	DX-52		coal workers' pneumoconiosis; moderate emphysema; minimal healed TB with coarse scar and probable small calcified granuloma
7-21-2000	EX-37 DX-52	Dr. Bodin	No evidence of acute cardiopulmonary process; tiny granuloma left upper lung
7-21-2000	EX-25 DX-52	Dr. Wheeler	Moderate hyperinflation lungs with decreased upper lung markings compatible with emphysema; small calcified granuloma below right apex is partly hidden; probably healed TB; negative for pneumoconiosis
7-21-2000	EX-26 DX-52	Dr. Scott ¹⁰	Negative for pneumoconiosis; calcified granulomata right apex and lateral left upper lung; hyperinflation lungs compatible with emphysema
7-25-2000	EX-37 DX-52	Dr. Bodin	Chronic obstructive pulmonary disease, possible small pleural effusions
7-25-2000	EX-27 DX-52	Dr. Wheeler	No silicosis or CWP; emphysema with moderate hyperinflation; 1 cm calcified granuloma in posterior inferior right apex and tiny calcified granuloma in posterolateral periphery left upper lobe and one in inferior lateral right lung compatible with healed TB more likely than healed histoplasmosis
7-25-2000	EX-28 DX-52	Dr. Scott	Negative for pneumoconiosis; hyperinflation lungs compatible with emphysema; calcified granulomata right apex, lateral left upper lung and lateral right lower lung
11-2-2000	EX-29 DX-52	Dr. Wheeler	No silicosis or CWP; moderate emphysema with hyperinflation; 1 cm calcified granuloma in posterior inferior right apex and tiny calcified granuloma in posterolateral periphery left upper lobe and one in inferior lateral right lung compatible

¹⁰ Dr. Scott is a "B" reader and Board-certified in radiology.

11-2-2000	EX-30 DX-52	Dr. Scott	with healed TB more likely than healed histoplasmosis Negative for pneumoconiosis; hyperinflation lungs compatible with emphysema; calcified granulomata right apex, lateral left upper lung and lateral right lower lung
11-15-2000	DX-44	Dr. Donner	Chronic obstructive pulmonary disease with bilateral parenchymal scarring; evidence of prior granulomatous disease; atherosclerosis of the thoracic aorta

B. CT Scan

Mr. Poole underwent a chest CT scan on September 29, 1999. (EX-23; DX-52). Dr. Wheeler interpreted the scan, opining that there was no evidence of pneumoconiosis. He found "moderate emphysema with hyperinflation lungs" and a "1.4-cm calcified granuloma in the posterior inferior apical portion" of the right upper lung, and a "3-mm calcified granuloma in posterolateral left upper lung compatible with healed tuberculosis." Dr. Scott also interpreted the CT scan finding a 1-cm calcified granuloma and linear scars in the posterior right upper lung; a small calcified granuloma in the lateral left upper lung; emphysema with hyperinflation and scattered bullous changes; and aortic and coronary artery calcification. He found no evidence of silicosis/coal workers' pneumoconiosis. (EX-24; DX-52). Dr. Sage found "no evidence of neoplasm involving the chest;" "parenchymal scarring and parenchymal calcification of calcified granuloma within the right upper lobe;" healed granulomatous disease of the chest;" and "lung changes consistent with chronic obstructive lung disease." (CX-16, p. 18).

C. Pulmonary Function Tests:

Date	EX. No.	Age/ Height	FEV ₁ ¹¹	FVC ¹²	MVV ¹³	Valid	Qualifies
8-20-1997	EX-32 ¹⁴	67/72"	0.84	1.98	28	—	yes
(post-bronchodilator)			0.87	2.31	—	—	yes

Interpretation: Moderate obstructive pulmonary impairment; possible restrictive ventilatory defect

¹¹ Forced expiratory volume in one second.
¹² Forced vital capacity.
¹³ Maximum voluntary ventilation.
¹⁴ EX-32; DX-52.

6-15-1998	EX-31 ¹⁵	68/71"	0.51	1.36	16	—	yes
(post-bronchodilator)			0.58	1.97	—	—	yes

3-15-1999	DX-11	69/69.5"	0.75	1.87	—	yes	yes
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Interpretation: Severe obstructive disease; restrictive disease cannot be excluded; post-bronchodilator not performed
 Found acceptable by Dr. Timothy Kennedy on July 28, 1999. (DX-14). Dr. Kennedy is board-certified in internal medicine, pulmonary diseases, and critical care medicine.

11-19-1999	DX-42	70/69.5"	0.52	2.02	18	—	yes
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Interpretation: Severe obstruction; moderate overinflation; diffusion capacity is mildly decreased; MVV is severely decreased.

D. Arterial Blood Gas Studies:

Test Date	Exhibit No.	pCO2	pO2	Qualifies	At rest/ After exercise
10-2-1997	EX-32	58.9	59.3	yes	At rest
	DX-52				
6-15-1998	EX-31	60	55	yes	At rest
	DX-52				
1-11-1999	EX-31	69	46	yes	At rest
	DX-52				
3-15-1999	DX-13	62.7	52.9	yes	At rest
Found valid by Dr. Timothy Kennedy on July 28, 1999. (DX-14)					
11-19-1999	DX-42	61	64	yes	At rest
11-15-2000	DX-44	130	93	yes	At rest

E. Medical Opinions:

On March 15, 1999, Mr. Poole was examined by Dr. Cullen A. Hebert. (DX-12; CX-11). He considered 15 years of underground coal mine employment, a history of smoking as much as three packs of cigarettes a day from the age of 35-40, a medical history, complaints of a productive cough and some wheezing, an x-ray, a pulmonary function study, a blood gas study, and a physical examination that revealed marked hyper-expansion of the chest. Dr. Hebert diagnosed chronic obstructive pulmonary disease of the emphysematous type due to smoking. He also found pneumoconiosis based on a chest x-ray and a history of working

¹⁵ EX-31; DX-52.

in a coal mine. Dr. Hebert did not address the miner's disability. Dr. Hebert provided a follow-up summary letter at Claimant's request on November 16, 2004, in which he further indicated Mr. Poole had a history of pneumoconiosis related to his work environment while coal mining. He noted Mr. Poole's chest x-ray showed the stigmata of COPD as well as fine nodular infiltrates mainly at the bases, consistent with pneumoconiosis. (CX-13). In an addendum on December 17, 2004, in response to Counsel for Claimant's specific inquiry, Dr. Hebert further opined Mr. Poole suffered from pneumoconiosis and that it was a substantial contributor to his death. Dr. Hebert further opined that Mr. Poole's pneumoconiosis likely caused the majority of his severe airways obstructive disease. (CX-15).

Mr. Poole treated with Dr. M. Laughlin Winkler from June 11, 1998 through June 19, 2000, presenting with a history of severe emphysema and COPD which Dr. Winkler attributed to his 15 years of coal mine work and 58 years of smoking. (CX-18, p. 1). On March 15, 1999, Dr. Winkler diagnosed Mr. Poole with COPD and emphysema. (CX-16, p. 13).

Records from North Oaks Hospital show that Mr. Poole presented on March 16, 1999 with chronic obstructive pulmonary disease with acute exacerbation and pneumonia. (DX-42; EX-31; DX-52). Dr. Winkler attended him and considered a medical history, a history of smoking half a pack of cigarettes a day for over sixty years, an x-ray, and the results of a physical examination, which showed wheezing and dullness to percussion. Dr. Winkler diagnosed severe end-stage emphysema with left lower lobe pneumonia, with basilar pneumonia, cor pulmonale with supraventricular tachycardia, and pulmonary insufficiency with oxygen saturation on room air at 89%. He admitted Mr. Poole to the hospital.

The March 18, 1999 discharge summary diagnosed left basilar pneumonia, pulmonary insufficiency secondary to chronic obstructive pulmonary disease, "multifocal atrial tachycardia," and degenerative arthritis. (DX-52; CX-16, pp. 33-34).

On September 7, 1999, Dr. Winkler again noted Mr. Poole was exposed to coal mines for 15 years and identified a history of "black lung." (CX-16, p. 11). On October 5, 1999, Dr. Winkler recommended that Mr. Poole stop smoking and reviewed a CT scan of his chest performed on September 29, 1999. Dr. Winkler identified the following impressions: "emphysema; black lung; SVT; and osteoarthritis." (CX-16, p. 9). On June 19, 2000, Dr. Winkler noted Mr. Poole continued to smoke one to one-half a

pack of cigarettes each day. He diagnosed Mr. Poole with acute emphysema and black lung, as well as chronic SVT, degenerative arthritis, and cigarette abuse. (CX-16, p. 8). In an opinion dated January 28, 2005, Dr. Winkler attributed Mr. Poole's death to "complications associated with severe end stage emphysema/COPD" and opined that the autopsy report "clearly demonstrates anthrasicosis in his lungs" and felt that anthrasicosis and cigarette smoking contributed to his death. (CX-18, p. 2).

Undated medical records from North Oaks Hospital Emergency Department indicate Mr. Poole presented with a past medical history of "black lung" and emphysema. (CX-16, p. 25). The medical records also note Mr. Poole had "end stage COPD" and "coal miner's lung." (CX-16, p. 26).

Dr. W. Brooks Emory, who is board-certified in Internal Medicine and the subspecialty of Pulmonary Diseases, examined Mr. Poole on November 19, 1999. (DX-42; DX-52). He considered a total of 23 years of underground coal mine employment, lastly as a machine operator, a medical history, symptoms of shortness of breath and a cough, as well as exertional dyspnea, and a history of smoking since the age of 19. Dr. Emory also took into account the results of a chest x-ray, pulmonary function study, a blood gas study, and a physical examination, which showed decreased breath sounds throughout all lung fields. He opined that the miner had pulmonary emphysema secondary to cigarette consumption and a physiological impairment of severe obstruction with hyperinflation and air trapping. He concluded that Mr. Poole did not have pneumoconiosis. He noted respiratory failure but did not address whether the claimant was totally disabled.

Dr. Emory later reviewed the medical records of his examination of the miner in a report dated June 19, 2000, at Employer's behest. (EX-2; DX-52). He explained that the November 19, 1999 chest x-ray was not consistent with pneumoconiosis, but compatible with emphysema, and that the "carboxy hemoglobin level" of the miner was consistent with active cigarette smoking. He opined, with medical certainty, that the miner's pulmonary impairment was strictly a consequence of his emphysema due to fifty years of smoking.

On June 13, 2000, Dr. Gregory J. Fino, who is board-certified in Internal Medicine and the subspecialty of Pulmonary Disease, reviewed the medical evidence of record. (EX-3; DX-52; CX-8). He considered the variously reported smoking histories, 13 years of coal mine employment, lastly as a continuous miner

operator, hospital records from September 1982 and March 1999, clinic records from August 1997 to April 1998, Dr. Winkler's records, the reports of Drs. Hebert and Emory, 21 x-ray reports, four pulmonary function studies, and five blood gas studies. Dr. Fino concluded that the evidence was insufficient to justify a diagnosis of simple coal workers' pneumoconiosis. In his opinion, the miner did not suffer from an occupationally acquired pulmonary condition. He found a disabling respiratory impairment due to smoking and ruled out coal mine dust inhalation as a cause of that impairment. Even assuming the presence of pneumoconiosis, Dr. Fino would not find that his pulmonary impairment was due in any part to that disease.

Dr. Fino provided a supplemental report dated December 13, 2001. (EX-4; DX-52). He reviewed five additional x-ray readings, office notes from March 15, 1999 to June 23, 1999, the March 16, 1999 discharge summary, the death summary, the death certificate, and the autopsy protocol. He found that the additional data further strengthened his opinion. He noted that the autopsy did not show evidence of pneumoconiosis. He pointed out that macules must be seen in order to diagnose the disease of pneumoconiosis, and even though the prosecutor found "slight" anthracosilicosis, she specified that no macules were seen. Dr. Fino opined the autopsy revealed the miner had "minimal to possibly no dust deposition" in his lungs. Thus, he concluded that coal mine dust exposure did not contribute to the miner's disabling respiratory impairment. He further asserted that the miner's respiratory death was due to smoking and was not caused, contributed to, or hastened by coal mine dust inhalation.

Mr. Poole was hospitalized at St. Tammany Parish Hospital from July 21, 2000 to July 27, 2000. (EX-37; DX-52). He was attended by Dr. Joseph Landers, who considered a "history of black lung," complaints of shortness of breath for a week and a cough, a history of smoking one-half pack of cigarettes a day for over 60 years, a medical history, an unspecified history as a coal miner, an x-ray, an EKG, and a physical examination, which showed diffuse end expiratory wheeze bilaterally and poor air movement. The discharge diagnoses were asthmatic bronchitis and a "history of black lung" secondary to working in the coal mines.

Mr. Poole was brought to North Oaks Hospital on November 15, 2000 and was seen by Dr. Susan Zacharia. (DX-44). He presented with severe respiratory distress and failure. Dr. Zacharia considered a history of chronic obstructive pulmonary disease, a chest x-ray, a blood gas study, and the results of a

physical examination. Mr. Poole died on the same day, and Dr. Zachaira's final diagnoses were respiratory failure, exacerbation of chronic obstructive pulmonary disease, and end-stage emphysema.

The death certificate was signed by Patricia Davidson, a registered nurse and coroner. (DX-35). She listed the cause of death as unspecified natural causes.

Following Mr. Poole's death, an autopsy limited to the chest was performed by Dr. Mina A. Gabrawy on November 21, 2000. (DX-36). Both a gross and a microscopic examination were made. Microscopically, Dr. Gabrawy found:

Display moderate congestion. Multiple alveoli display hemosiderin laden macrophages. Sections of the upper lobes display dilated air sacs with floating septa. Bronchi display focal infiltrates by neutrophils with fibrin enmeshed neutrophils partially filled the lumina of multiple bronchi and infiltrate the adjacent pulmonary parenchyma with areas of alveolar hemorrhage and fibrin deposition. Small amount of dark gray to black and brown pigment is deposited within alveolar septa. No macules are seen. The granuloma noted in sections of the right hilar node displays central caseation.

The final diagnoses were: (1) coronary atherosclerosis, marked; (2) pulmonary congestion, marked; (3) bilateral bronchopneumonia; and (4) slight anthracosilicosis, although no macules were seen.

Dr. Mina Gabrawy, a board-certified pathologist, was deposed on August 19, 2004. (EX-52). Dr. Gabrawy estimated he has performed autopsies on 25 to 30 coal miners. (EX-52, pp. 7-8). Dr. Gabrawy described the usual procedures followed in performing an autopsy and testified that, although the body is not usually embalmed at the time of autopsy, as here, the presence of embalming fluid did not affect the results in the present matter. (EX-52, pp. 10-13, 24). Dr. Gabrawy testified that he typically looks for the following criteria in diagnosing black lung disease: the consistency and color of the lungs, the presence of black ink-like fluid when the lungs are cut, black lymph nodes, and the presence of macules and fibrosis. (EX-52, pp. 14-15). According to Dr. Gabrawy, the presence of macules is a definite criterion in diagnosing black lung disease. (EX-52, pp. 15-16). In the present matter, he found nothing to

indicate the presence of coal workers' pneumoconiosis, noting that the color of Mr. Poole's lungs was gray and that no macules were found. He found no evidence of pneumoconiosis either microscopically or upon gross examination. (EX-52, pp. 16-17).

Dr. Gabrawy noted black pigment in the walls of alveoli, which he described as "anthracosilicosis" in the autopsy report. He testified the black pigment indicated that Mr. Poole worked in coal mines, but that discoloration could also result from cigarette smoking. (EX-52, pp. 17-19) Dr. Gabrawy testified that he did not find actual tissue damage and that the diagnosis of anthracosilicosis is not equivalent to a diagnosis of coal workers' pneumoconiosis. (EX-52, pp. 18-19). Because he found no evidence of disease as a result of coal dust inhalation, Dr. Gabrawy does not believe coal dust inhalation impaired Mr. Poole's lung function; however, he stated his findings would have to be correlated with other medical records and physical examinations performed prior to Mr. Poole's death. (EX-52, pp. 21-22).

Dr. Gabrawy found evidence of heart disease and opined that Mr. Poole's death was caused by coronary artery disease. (EX-52, pp. 19-23). Dr. Gabrawy further opined that Mr. Poole did not have coal workers' pneumoconiosis, and, thus, it did not cause or hasten his death. (EX-52, pp. 22-23).

Dr. John P. Kress is board-certified in Internal Medicine and pulmonary medicine subspecialty and is an Assistant Professor of Medicine, Section of Pulmonary and Critical Care, at the University of Chicago. Dr. Kress reviewed medical evidence and provided a report dated December 20, 2001, at the behest of Employer. (EX-5; DX-52). He considered 14 years of coal mine employment, a 52-year smoking history, a medical history, several x-ray readings, four pulmonary function studies, a blood gas study, a chest CT scan reading, Dr. Hebert's report, hospital records, and the autopsy report. He opined that the miner suffered from severe emphysema from longstanding tobacco abuse. While he recognized an association between coal dust exposure and emphysema, he relied upon the autopsy which showed no evidence of the characteristic lesion of coal workers' pneumoconiosis-centriacinar emphysema in combination with coal macules-to conclude that Mr. Poole did not suffer from coal workers' pneumoconiosis. Dr. Kress specified that he believed the autopsy prosector's diagnosis of "slight anthrasilicosis" was an incidental finding because she did not find any coal macules, and there was no mention of interstitial fibrosis, coal nodules, muscular pulmonary arteries surrounded

by coal dust, subpleural dust deposits, hilar or mediastinal lymph node enlargement, or tattooing of the parietal lymphatic channels by coal dust. Likewise, he found no evidence to suggest that pneumoconiosis hastened, caused, or contributed to the miner's death. Dr. Kress is board-certified in internal medicine, pulmonary medicine, and critical care medicine.

At the request of the Department of Labor, Dr. Cecile Rose provided a report dated March 9, 2004, after reviewing the pertinent regulations, reports of Mr. Poole's medical examination, medical evidence review and report by Dr. Fino, and the death certificate and autopsy report.¹⁶ Dr. Rose noted that Mr. Poole spent 14 years as a coal miner and had a "long history" of cigarette smoking, which she testified was in excess of 50 pack-years. (CX-4, p. 1; CX-1, p. 17). Dr. Rose opined to a reasonable degree of medical certainty that Mr. Poole had pneumoconiosis and that his exposure to coal mine dust probably substantially aggravated and was significantly related to his severe pulmonary impairment. (CX-4, p. 1). She further opined that pneumoconiosis "substantially contributed to his total disability by materially affecting his pulmonary condition and materially worsening his pulmonary impairment." She identified tobacco smoking as another contributing factor to Mr. Poole's obstructive impairment. (CX-4, p. 2). Dr. Rose further opined Mr. Poole died a respiratory death caused by severe underlying obstructive lung disease and emphysema to which his coal mine dust exposure substantially contributed. She opined to a reasonable degree of medical probability that Mr. Poole's death was "hastened by his underlying pneumoconiosis and obstructive lung disease and that this pneumoconiosis was a substantially contributing factor leading to his death." (CX-4, p. 2).

Dr. Rose testified that she did not review Mr. Poole's x-rays, but found "variability" among the interpretations regarding the presence of nodular pneumoconiosis; nonetheless, she testified that coal workers' pneumoconiosis can be present in the absence of a positive chest x-ray read. (CX-1, pp. 21-22). She opined that Mr. Poole's pulmonary function tests revealed an obstructive, and not restrictive, abnormality. (CX-1, p. 22). While she agreed that Mr. Poole's cigarette smoking substantially contributed to his obstructive lung impairment and

¹⁶ Dr. Rose is board-certified in pulmonary medicine, occupational and preventive medicine, and internal medicine. Dr. Rose is the Director, Occupational Medicine Program, Division of Environmental and Occupational Health Sciences, National Jewish Medical and Research Center and Associate Professor of Medicine and Preventive Medicine and Bionetics, University of Colorado School of Medicine. (CX-1, p. 10; EX-51).

probably played a more significant role than dust exposure, she could not quantify a percentage contribution of coal dust exposure and tobacco smoke exposure. (CX-1, pp. 23-24). Nonetheless, she opined that both coal dust exposure and cigarette smoke exposure played a substantial contributing role in Mr. Poole's lung disease and death. (CX-1, p. 31).

Dr. Rose indicated that smoking the equivalent of 50 pack-years can result in emphysema in people who have not been coal miners. However, she also testified that 14 years of exposure to coal mine dust alone could be sufficient, depending on the concentrations of exposure, to cause the degree of obstructive lung impairment seen in Mr. Poole. (CX-1, p. 39).

Dr. Rose further testified that, despite the absence of "coal macules," the autopsy findings of anthracosilicosis and probable emphysema can be contributed to or aggravated by exposure to coal mine dust. (CX-1, pp. 34-35). Additionally, she testified that emphysema without coal macules can be present in people who have "coal-mine-dust-aggravated emphysema," and that the absence of coal macules and fibrosis in an autopsy report does not "rule out" the likelihood that Mr. Poole's death was contributed to or hastened by exposure to coal mine dust. (CX-1, p. 36).

Dr. Emory reviewed x-ray interpretations, records of hospitalization, documentation from Employer, pulmonary function studies, as well as his own medical records from a January 19, 1999 examination in preparation for a July 18, 2004 report submitted at the request of Employer. He concluded that the "overwhelming medical opinions" and absence of coal maculae on the autopsy report do not support the diagnosis of coal worker's pneumoconiosis and he disagreed with the interpretations and opinions of Dr. Rose, which are "not in keeping with the thinking of the other experts." (EX-46). Dr. Emory reviewed Mr. Poole's x-ray reports in preparation of an August 23, 2004 report, in which he maintained there was no evidence that Mr. Poole's death was a consequence of coal workers' pneumoconiosis and that death was "secondary to the progression of severe pulmonary emphysema and infection." In rendering his opinion, Dr. Emory noted that the radiographs reviewed by "B" Reader criteria failed to show pneumoconiosis, while only one interpretation by an "unknown 'B' reader" found the presence of pneumoconiosis. (EX-47). He maintained his opinions in a supplemental report dated January 14, 2005 and expressed disagreement with the opinions of Dr. Hebert regarding

contribution of pneumoconiosis to the progressive pulmonary deterioration and death of Mr. Poole. (EX-56).

On August 19, 2004, Dr. Fino prepared a report at Employer's request after reviewing his previous reports; additional pulmonary function studies; a 1997 exercise test; chest x-ray and CT scan readings; emergency department and hospital admission records dated July 21, 2000 through July 27, 2000; the transcript of the January 14, 2002 hearing; and the report of Dr. Rose. He disagreed with Dr. Rose's opinion that coal mine dust exposure substantially contributed to Mr. Poole's severe underlying obstructive lung disease and emphysema. Although he agreed that 15 years is sufficient exposure to develop coal workers' pneumoconiosis, he would not expect an "obstructive abnormality" of such a significant and disabling nature. He further agreed with Dr. Rose's opinion that "someone with severe lung disease such as [Mr. Poole] may not be able to survive any type of insult as well as someone without lung disease." Nonetheless, Dr. Fino concluded that Mr. Poole's condition was due to cigarette smoking rather than coal mine dust exposure and that coal mine dust inhalation did not cause, contribute to, or hasten his death. (EX-48).

In rendering his opinion, Dr. Fino cited research by Attfield and Hodus that studied the decline in "FEV1" in coal mine workers who were smokers and non-smokers. The study perceived an average loss of "2-3cc per year of work" with the current dust regulations. Dr. Fino indicated that a decrease of "5-9cc per year of work" would be expected for work performed before the dust regulations. Based on the study, Dr. Fino concluded that 135cc of FEV1, at most, would have been lost as a result of Mr. Poole's coal mine employment, assuming he was exposed to pre-dust regulation levels for his entire career. According to Dr. Fino, this is an insignificant amount and Mr. Poole would have been as disabled and impaired without the estimated loss of 135cc of FEV1. (EX-48, pp. 6-7).

After reviewing Dr. Hebert's November 16, 2004 letter and its addendum, Dr. Fino provided an additional opinion on January 20, 2005, in which he disagreed with the opinions of Dr. Hebert. Specifically, Dr. Fino opined that Mr. Poole's chest x-ray revealing "fine nodular infiltrates mainly at the base" is not consistent with coal workers' pneumoconiosis as Dr. Hebert had indicated. Dr. Fino noted that coal mine dusts may cause "abnormalities" to appear on chest x-rays. He described the abnormalities as "rounded opacities" which are found in particular locations of the lung; namely, the opacities would

first appear in the upper portion of the right lung and lastly would appear in the two "lower zones." According to Dr. Fino, the presence of "irregular opacities" in the absence of "rounded opacities" is inconsistent with the diagnosis of coal workers' pneumoconiosis. (EX-57, pp. 1-2).

Regarding emphysema and coal dust exposure, Dr. Fino first noted that "emphysema" has a "pathological and a clinical meaning."¹⁷ While a clinical diagnosis of emphysema does not always occur when pathological emphysema is present, clinical emphysema will always have a "pathological correlate." Dr. Fino addressed the issue of whether or not simple coal workers' pneumoconiosis or coal mine dust inhalation alone causes clinically significant emphysema, noting that the presence of emphysema in the lungs does not automatically mean a respiratory impairment is present. Dr. Fino quoted "Dr. Parkes'" statement that "there is no convincing evidence to support the view that disabling emphysema other than irregular or scar emphysema associated with some cases of PMF, is more common in coal miners than in the population at large." Dr. Fino also quoted the Surgeon General's opinion that the kind of emphysema seen in coal miners is "best referred to as focal dust emphysema," which does not pose an increased risk of mortality to the miner. (EX-57, pp. 3-4). Dr. Fino concluded that the objective medical evidence showed a smoking-related obstruction and that Mr. Poole did not have a coal mine dust-related pulmonary condition that was a contributing or hastening cause of his death. (EX-57, pp. 4-5).

Dr. Kress reviewed additional chest radiograph reports, the memorandum to Dr. Rose, the report of Dr. Rose, and the hearing transcript, in preparation of an August 31, 2004 report at the request of Employer. He maintained the opinion that Mr. Poole did not have coal workers' pneumoconiosis, noting that six of eleven radiographs were unequivocally read as showing no evidence of pneumoconiosis. Of the remaining five radiographs, only one reading identified the possibility of granulomatous disease as well as pneumoconiosis. Nonetheless, based on the finding of "unequivocal granulomatous disease" on autopsy and no evidence of findings of macules or any of the other findings typically seen in coal miners pneumoconiosis, Dr. Kress did not believe the evidence supported a finding of coal workers' pneumoconiosis. (EX-49).

¹⁷ Pathological emphysema is the dilation of the air sacs in the lungs. A clinical diagnosis of emphysema means an individual experiences shortness of breath due to the dilation of the air sacs and the resulting "obstructive ventilatory abnormality." (EX-57, p. 2).

Dr. Kress also opined that emphysema in Mr. Poole was caused by cigarette smoking and was not caused by coal dust. According to Dr. Kress, "[i]f his coal dust exposure substantially aggravated and was significantly related to his severe pulmonary impairment, we must conclude that his coal dust exposure caused his severe emphysema." He further stated it would be "highly implausible" to attribute coal dust exposure to the degree of emphysema found in Mr. Poole, in the absence of findings of coal macules, interstitial fibrosis, coal nodules, and other typical findings in instances of coal workers' pneumoconiosis. (EX-49).

In a supplemental letter dated January 18, 2005, Dr. Kress noted that he reviewed the letters of Dr. Hebert dated November 16, 2004 and December 17, 2004. Dr. Kress maintained the opinions expressed in his August 31, 2004 report and "strongly" disagreed with Dr. Hebert's opinion that Mr. Poole's obstructive lung disease was caused most likely by pneumoconiosis. (EX-55).

DISCUSSION

Miner's Claim

Applicable Law:

The claim was made after March 31, 1980, the effective date of 20 C.F.R. Part 718, and must therefore be adjudicated under those regulations. To establish entitlement to benefits under Part 718, claimant-miner must establish, by a preponderance of the evidence, that he is totally disabled due to pneumoconiosis arising out of coal mine employment. See 20 C.F.R. §§ 719.3, 718.202, 718.203, 718.204; Gee v. W.G. Moore, 9 B.L.R. 1-4, 1-5 (1986); Roberts v. Bethlehem Mines Corp., 8 B.L.R. 1-211, 1-212 (1985). Failure to establish any of these elements precludes entitlement. Anderson v. Valley Camp of Utah, Inc., 12 B.L.R. 1-111, 1-112 (1989); Trent v. Director, OWCP, 11 B.L.R. 1-26, 1-27 (1987).

Pneumoconiosis:

In establishing entitlement to benefits, claimant must initially prove the existence of pneumoconiosis under 20 C.F.R. § 718.202. Claimant has the burden of proving the existence of pneumoconiosis, as well as every element of entitlement, by a preponderance of the evidence. See Director, OWCP v. Greenwich

Collieries, 512 U.S. 267 (1994). Pneumoconiosis is defined by the regulations at § 718.201:

(a) For the purpose of the Act, "pneumoconiosis" means a chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment. This definition includes both medical, or "clinical," pneumoconiosis and statutory, or "legal," pneumoconiosis.

(1) Clinical Pneumoconiosis. "Clinical pneumoconiosis" consists of those diseases recognized by the medical community as pneumoconioses, i.e., the conditions characterized by permanent deposition of substantial amounts of particulate matter in the lungs and the fibrotic reaction of the lung tissue to that deposition caused by dust exposure in coal mine employment. This definition includes, but is not limited to coal workers' pneumoconiosis, anthracosilicosis, anthracosis, anthrosilicosis, massive pulmonary fibrosis, silicosis or silicotuberculosis, arising out of coal mine employment.

(2) Legal Pneumoconiosis. "Legal pneumoconiosis" includes any chronic lung disease or impairment and its sequelae arising out of coal mine employment. This definition includes, but is not limited to, any chronic restrictive or obstructive pulmonary disease arising out of coal mine employment.

(b) For purposes of this section, a disease "arising out of coal mine employment" includes any chronic pulmonary disease or respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust exposure in coal mine employment.

(c) For purposes of this definition, "pneumoconiosis" is recognized as a latent and progressive disease which may first become detectable only after the cessation of coal mine dust exposure.

20 C.F.R. § 718.201 (2004).

Section 718.202(a) sets forth four methods for determining the existence of pneumoconiosis.

(1) Under § 718.202(a)(1), a finding that pneumoconiosis exists may be based upon x-ray evidence.

In this case, there are 34 readings of 13 separate x-rays, along with three separate readings of one CT scan. Five of the x-ray readings are by "B" readers, whose interpretations merit great weight. Nineteen of the x-ray readings and two of the CT scans are by physicians who are both board-certified radiologists and "B" readers. Thus, their readings are entitled to even greater weight. Scheckler v. Clinchfield Coal Co., 7 BLR 1-128 (1984). There are a total of three positive readings.

The first x-ray was taken on September 28, 1982 and was not found to be positive for pneumoconiosis. The film was not reviewed again.

The next two x-rays were taken August 25, 1997 and October 2, 1997. The original interpreter of each, Dr. Kerber, did not make a finding of pneumoconiosis. The films were also read by Dr. Main and Dr. Wheeler, both of whom are "B" readers and also board-certified radiologists. Both physicians specifically found the x-rays negative for pneumoconiosis. Based on the superior credentials of Drs. Main and Wheeler, I consider these two x-rays negative.

The June 15, 1998 x-ray was interpreted by Dr. Williams, who has no particular credentials for reading x-rays. He did not diagnose pneumoconiosis. Dr. Wheeler and Dr. Worrell, both dually certified readers, interpreted the x-ray as negative for pneumoconiosis. Based on their excellent qualifications, I find this x-ray negative.

The March 15, 1999 x-ray was originally read by Dr. Preger, a "B" reader. She not only found category two pneumoconiosis, but also complicated pneumoconiosis in the form of size A large opacities. She commented, however, that the appearance was atypical for coal workers' pneumoconiosis. However, in Cranor v. Peabody Coal Co., 22 BLR 1-1 (1999), the Benefits Review Board held that an interpreting physician's comment that pneumoconiosis found on x-ray was not coal workers' pneumoconiosis did not affect her diagnosis of the disease under § 718.202(a)(1), "but merely addresses the source of the diagnosed pneumoconiosis." Therefore, I consider Dr. Preger's reading positive for the disease. While Dr. Wheeler, a dually certified reader, read the film as negative, Dr. Worrell, an equally qualified radiologist and "B" reader, confirmed Dr.

Preger's positive diagnosis. Consequently, I consider this particular x-ray positive for pneumoconiosis.

The March 16, 1999 x-ray was read by Dr. Wheeler as negative but by Dr. Worrell as positive, although he found a somewhat lesser degree of the disease than in the previous x-ray. Dr. Williams found the film positive for COPD, but was silent regarding the presence of pneumoconiosis. Because the evidence is equally divided concerning this particular x-ray, I consider it negative.

The March 20, 1999 x-ray was read by Dr. Miller, who did not diagnose pneumoconiosis. Drs. Worrell and Wheeler read the film and both found it negative for the disease. Consequently, I find this film negative.

Dr. Wester, whose credentials are not of record, interpreted the September 13, 1999 x-ray and did not find it showed pneumoconiosis. Dr. Wheeler read the film and confirmed that it was negative for pneumoconiosis. Therefore, I find this x-ray negative.

The November 19, 1999 x-ray was interpreted by two "B" readers, Drs. Hippensteel and Castle, and by two dually certified readers, Drs. Matthews and Wheeler. All four agreed that the film was negative for pneumoconiosis. Consequently, I adopt their findings.

The July 21, 2000 and July 25, 2000 x-rays were each read by a hospital radiologist, Dr. Bodin, whose credentials are not of record. He did not diagnose pneumoconiosis. Both films were read by Drs. Wheeler and Scott, both of whom are dually certified readers. Both Dr. Wheeler and Dr. Scott found the two x-rays negative for pneumoconiosis. Thus, I also consider this x-ray negative.

The November 2, 2000 x-ray was read by Drs. Wheeler and Scott, who are dually certified readers, as negative for pneumoconiosis. Therefore, I find this x-ray to be negative.

The final x-ray, dated November 15, 2000, was read by Dr. Donner, whose qualifications are not of record. His interpretation did not include a diagnosis of pneumoconiosis. I, therefore, consider this final x-ray negative.

I also note that Mr. Poole underwent a CT scan on September 29, 1999. It was interpreted by Dr. Wheeler, Dr. Scott, and Dr. Sage, who found no evidence of pneumoconiosis.

In summary, there are three positive readings and 31 negative readings. The three positive readings come from well-qualified interpreters. However, the overwhelming number of the most highly qualified readers found the x-rays negative. Furthermore, although I found the March 15, 1999 hospital x-ray positive, the seven more recent x-rays were negative, including one x-ray taken just a day after the March 15, 1999 x-ray and another taken just five days later. Moreover, the six most recent x-rays were unanimously found negative. Furthermore, the CT scan was found negative for pneumoconiosis. Based on the above analysis, the majority of readings by the best-qualified interpreters, and the most recent x-ray evidence, I find that the x-ray evidence overwhelmingly does not support a finding of coal workers' pneumoconiosis pursuant to § 718.201(a). Goss v. Eastern Associated Coal Co., 7 B.L.R. 1-400 (1984).

(2) A biopsy or autopsy conducted and reported in compliance with 20 C.F.R. § 718.106 may also be the basis for finding the existence of pneumoconiosis. 20 C.F.R. § 718.202(a)(2). While no biopsy was performed, a partial autopsy, limited to the lungs, was performed.

Dr. Gabrawy, who performed the limited autopsy, diagnosed anthracosilicosis despite not finding any coal macules either grossly or microscopically. No other physician viewed the autopsy slides, but Dr. Fino, Dr. Kress, Dr. Emory, Dr. Winkler, and Dr. Rose reviewed the autopsy protocol.¹⁸ Dr. Fino and Dr. Kress pointed out that pathologically, because no macules were seen, the diagnosis of anthracosilicosis could not properly be made. Dr. Emory also noted the absence of "maculae," which he described as "the classic histological microscopic finding of coal worker's pneumonociosis." Accordingly, he concluded the autopsy failed to show characteristic features of pneumoconiosis.

However, Dr. Rose opined "to a reasonable degree of medical certainty" that Mr. Poole did have pneumoconiosis based on her review of the medical evidence. She testified that she would "not necessarily" expect to see coal macules in Mr. Poole's

¹⁸ The record evidence indicates a copy of the autopsy protocol was forwarded to Dr. Hebert, but neither his November 16, 2004 report nor its addendum reference a review of the autopsy information.

lungs because emphysema can be present without coal macules when there is "coal-mine-dust-aggravated emphysema." She noted that findings within alveoli were consistent with emphysema related to coal mine dust exposure and further noted that a finding of "slight anthracosilicosis" and "probable emphysema" can be contributed to or aggravated by exposure to coal mine dust.

While Dr. Rose possesses excellent credentials given her certifications and work within the area of occupational lung disease, I find that Drs. Fino, Kress, and Emory provided more cogent and reasoned opinions as noted above. Also, I find the testimony of Dr. Gabrawy provides corroborative support for the opinions of Drs. Fino, Kress, and Emory.

I find the testimony of Dr. Gabrawy to be the most persuasive regarding the autopsy evidence. It is reasonable to assign greater weight to the opinion of the physician who performed the autopsy over the opinions of others who reviewed only findings without reviewing the slides. Terlip v. Director, OWCP, 8 BLR 1-363 (1985); Fetterman v. Director, OWCP, 7 BLR 1-688 (1985). Despite Dr. Gabrawy's statement that macules were absent, he felt that the gross and microscopic examinations of Mr. Poole's lung tissue warranted a diagnosis of anthracosilicosis, which is included in the definition of pneumoconiosis. 20 C.F.R. § 718.201. However, during his deposition, Dr. Gabrawy testified that he used the term "anthracosilicosis" to describe black pigmentation found in the walls of alveoli; he did not diagnose coal workers' pneumoconiosis because he found no evidence of disease resulting from coal dust inhalation. He further testified that he found no evidence of pneumoconiosis on gross or microscopic examination. I choose to rely on Dr. Gabrawy's testimony and thus, find that the autopsy evidence does not support a finding of pneumoconiosis. Hapney v. Peabody Coal Co., 22 B.L.R. 1-106 (2001) (en banc) (The Board noted that 20 C.F.R. §718.202(a)(2)(2001) contained an amendment to the prior version of the regulation "to add that a finding on autopsy or biopsy of anthracotic pigmentation shall not be sufficient, by itself, to establish the existence of pneumoconiosis).

(3) Section 718.202(a)(3) provides that pneumoconiosis may be established if any one of several cited presumptions are found to be applicable. Section 718.305 is not applicable to claims filed after January 1, 1982. The presumption of § 718.306 is applicable only in a survivor's claim filed prior to June 30, 1982. Under 20 C.F.R. § 718.304, there is an irrebuttable presumption that a miner is totally disabled due to

pneumoconiosis if he is suffering from a chronic dust disease of the lung, which, when diagnosed by x-ray, yields one or more large opacities and would be classified in Category A, B, or C.

In this case, Dr. Preger, a "B" reader, found size A large opacities on the March 15, 1999 x-ray. She commented, however, that the appearance was atypical for coal workers' pneumoconiosis. Furthermore, among the other 33 x-ray readings, no other physician found large opacities. Dr. Worrell, who also found the March 15, 1999 x-ray positive for pneumoconiosis, did not find any large opacities, and he is both a "B" reader and a board-certified radiologist. Because I find that even Dr. Preger questioned the existence of large opacities due to pneumoconiosis, Dr. Worrell did not make such a finding on the same x-ray, and no other physicians suggested the presence of complicated pneumoconiosis, I conclude that the evidence does not establish the existence of complicated pneumoconiosis. Therefore, the claimants are not entitled to the irrebuttable presumption of § 718.304, and the evidence does not establish pneumoconiosis under subsection (a)(3).

(4) The fourth and final way in which it is possible to establish the existence of pneumoconiosis under § 718.202 is set forth in subsection (a)(4) which provides in pertinent part:

A determination of the existence of pneumoconiosis may also be made if a physician, exercising sound medical judgment, notwithstanding a negative x-ray, finds that the miner suffers or suffered from pneumoconiosis as defined in § 718.201. Any such finding shall be based on electrocardiograms, pulmonary function studies, physical performance tests, physical examination, and medical and work histories. Such a finding shall be supported by a reasoned medical opinion.

This section requires a weighing of all relevant medical evidence to ascertain whether or not claimant has established the presence of pneumoconiosis by a preponderance of the evidence. Any finding of pneumoconiosis under § 718.202(a)(4) must be based upon objective medical evidence and also be supported by a reasoned medical opinion. A reasoned opinion is one which contains underlying documentation adequate to support the physician's conclusions. Fields v. Island Creek Coal Co., 10 B.L.R. 1-19, 1-22 (1987). Proper documentation exists where the physician sets forth the clinical findings, observations, facts,

and other data on which he bases his diagnosis. Oggero v. Director, OWCP, 7 B.L.R. 1-860 (1985).

Dr. Hebert and Dr. Rose diagnosed pneumoconiosis. Dr. Landers observed a "history of black lung." The records from Dr. Zacharia do not address the presence or absence of pneumoconiosis. The records from Dr. Winkler noted a history of "black lung" and, after reviewing the autopsy report, he concluded anthracosilicosis and cigarette smoking contributed to Mr. Poole's death. Drs. Emory, Fino, Kress, and Gabrawy opined that Mr. Poole did not suffer from pneumoconiosis. Several factors affect the weight I place on these opinions.

I place little weight on Dr. Hebert's March 19, 1999 opinion, as the Benefits Review Board determined that it did not "state a meaningful diagnosis of Mr. Poole's lung disease under 20 C.F.R. §718.202(a)(4) and because it completely failed to address the relationship, if any, between Mr. Poole's lung disease and total disability." However, I place value on Dr. Hebert's November 16, 2004 opinion and the December 17, 2004 addendum, despite his reliance on the earlier report in rendering these additional opinions. I find the November 16, 2004 report to be more reliable than the earlier report because Dr. Hebert more specifically set forth that Mr. Poole presented with a chest x-ray consistent with pneumoconiosis and attributed the presence of pneumoconiosis to Mr. Poole's exposure to coal dust. The x-ray he relied upon was found positive by a "B" reader and later confirmed positive by a "B" reader who is also a board-certified radiologist; thus, I find the reliance on the x-ray to be reasonable.¹⁹

However, I question the accuracy of the smoking history considered by Dr. Hebert. The March 15, 1999 report reflected a smoking history of 29-30 years, yet other records indicate a smoking history of twice that much. Stark v. Director, OWCP, 9 BLR 1-36 (1986). The subsequent reports considered a "history of smoking," but do not address whether Dr. Hebert sought clarification of the extent of Mr. Poole's smoking history. Accordingly, the amount of weight I afford to Dr. Hebert's opinions is diminished for this reason.

I place no weight on the diagnosis by Dr. Landers of a "history of black lung" because it is clear from the hospital records that this finding was based on the history provided to him by the miner himself. Neither of the two x-rays taken during

¹⁹ The x-ray is identified in the March 15, 1999 report.

the hospitalization when Dr. Landers attended the miner was read as positive for pneumoconiosis. Thus, Dr. Landers's diagnosis of a "history" of black lung lacks objective medical support and reliability.

A review of Dr. Winkler's reports reveals no objective findings or diagnostic testing upon which a diagnosis of "black lung" could be founded. Rather, Dr. Winkler's notes of September 1999 indicated for the first time that Mr. Poole provided a history of "black lung." A subsequent chest x-ray revealed a "questionable mass," but Dr. Winkler did not specifically opine that pneumoconiosis was present in either the chest x-ray or a chest CT scan. Finally, his January 28, 2005 opinion that anthracosilicosis contributed to Mr. Poole's demise was based upon Dr. Winkler's review of the autopsy report.

Because there is a lack of objective medical evidence and supporting "documentation," I find Dr. Winkler has not offered a well-reasoned opinion. As his diagnosis of "black lung" arguably is based solely upon patient history, I afford no weight to the diagnosis. Similarly, I afford no weight to his conclusion that anthracosilicosis contributed to Mr. Poole's demise, in light of the contradictory testimony of Dr. Gabrawy.²⁰

Dr. Rose opined that Mr. Poole suffered from pneumoconiosis and that pneumoconiosis was a substantial contributor to his death. In preparation of her report, Dr. Rose reviewed and relied on interpretations of Mr. Poole's x-rays, along with the autopsy report and other information. While the weight to be afforded Dr. Rose's opinion arguably could be lessened due to her failure to review any actual x-rays, I decline to do so because the other consultative doctors based their opinions on review of x-ray reports and interpretations, as well. I also find that Dr. Rose's credentials are at least the equivalent of the other physicians who rendered opinions in this matter.

Nonetheless, I do not find that the entirety of Dr. Rose's opinion is well-reasoned and supported. In forming her conclusions regarding the existence of pneumoconiosis in Mr. Poole, Dr. Rose noted "variability" among the interpretations of the x-rays; yet, she did not address the overwhelming number of interpretations that found the x-rays did not exhibit coal

²⁰ As previously discussed, Dr. Gabrawy testified that the diagnosis of anthracosilicosis was based simply upon the presence of discoloration in the alveoli and that there was no evidence of disease as a result of coal dust inhalation.

workers' pneumoconiosis. Rather, Dr. Rose dismissed the negative readings by simply stating that coal workers' pneumoconiosis can be present in the absence of a positive chest x-ray reading. Dr. Rose did not provide any authoritative documentary support for her statement, nor did she offer an explanation of how she determined that pneumoconiosis was present in light of the materials she actually reviewed.

However, in her report dated March 9, 2004, Dr. Rose noted Mr. Poole's history as a coal miner; interpreted the results of Dr. Hebert's clinical evaluation of Mr. Poole in March 1999 as showing severe obstructive lung disease; and indicated Mr. Poole's "pulmonary function and arterial blood gas findings showed him to be severely impaired." These findings, combined with the information contained in the autopsy report, resulted in Dr. Rose's opinion that Mr. Poole did have pneumoconiosis at the time of his death. Although I find Dr. Rose provides some support for her opinion in the March 2004 report, I also note that her conclusions based on the autopsy report are arguably in contradiction with the conclusions and testimony of Dr. Gabrawy. I find the existence of conflicting opinions detracts from the weight of Dr. Rose's report, as Dr. Gabrawy actually performed the autopsy and generated the autopsy report based on first hand examination. As Dr. Rose did not offer an explanation as to how she arrived at a contrary conclusion, I do not give persuasive weight to her opinion.

Based on the foregoing, I afford some probative weight to Dr. Rose's opinion, given her credentials and her review of medical evidence. However, I do not give her opinion persuasive weight in light of her arguably conclusory statements regarding the x-rays and in light of her interpretation of the autopsy report which differs from the contradictory findings of the prosector.

I place greater weight on the opinions of Dr. Emory and Dr. Kress because the opinions are well-documented and reasoned. Perry v. Director, OWCP, 9 BLR 1-1 (1986). The x-ray on which Dr. Emory originally relied was found negative by two "B" readers and two dually certified readers. I note that he considered an exaggerated coal mine employment history, but since this error could only have aided the miner's claim, I find that it does not detract from the credibility of Dr. Emory's opinion. In his later reports, Dr. Emory maintained his opinion that Mr. Poole did not suffer from coal workers' pneumoconiosis. He supported his opinion by noting that no "B" readers identified pneumoconiosis in the x-rays and that only one

interpretation, by an "unknown 'B' reader," Dr. Worrell, found pneumoconiosis. Dr. Emory also specifically disagreed with Dr. Rose's interpretation of the medical evidence and found "no antemortem or postmortem evidence of coal worker's pneumoconiosis . . ."

Similarly, Dr. Kress initially opined Mr. Poole did not suffer from pneumoconiosis because the autopsy report showed no evidence of coal macules or characteristic lesions. He believed the diagnosis of "slight anthracosilicosis" was an incidental finding due to the absence of coal macules and other findings characteristic of pneumoconiosis. I find his initial opinion is supported by the deposition testimony of Dr. Gabrawy. In his subsequent reports, Dr. Kress noted that six of eleven radiographs were read as negative for pneumoconiosis. He further noted that one reading identified possible granulomatous, which was found on autopsy.

The opinion of Dr. Fino also merits greater weight because it is based on a review of medical evidence which provided him with a broad base of information from which to draw his conclusions. I find Dr. Fino provided a well-reasoned opinion supported by the documentation contained within Mr. Poole's medical records, as well as by the studies cited within his reports.

The conclusions set forth by Drs. Emory, Kress, and Fino are supported by the overall weight of the x-ray evidence and the examining physicians' clinical findings. Consequently, I find their opinions well-documented and reasoned. Moreover, Drs. Kress and Fino possess superior qualifications in the field of pulmonary medicine. Scott v. Mason Coal Co., 14 BLR 1-38 (1990). Accordingly, I afford greater weight to the opinions of Drs. Emory, Kress, and Fino.

I find the opinions of Drs. Emory, Fino, and Kress more persuasive than the opinions of Dr. Hebert, Dr. Winkler, Dr. Rose, and, to the extent it is an opinion of pneumoconiosis, the conclusions of Dr. Landers. Therefore, I conclude that the credible medical opinion evidence does not tend to establish the existence of pneumoconiosis pursuant to §718.202(a)(4).

Moreover, consideration of all the evidence under Section 718.202 also leads to the conclusion that the claimant has failed to establish the existence of pneumoconiosis by a preponderance of the evidence. Island Creek Coal Co. v. Compton, 211 F.3d 203, 22 BLR 2-162 (CRT)(4th Cir. 2000). After reviewing

the deposition of Dr. Gabrawy, I find the autopsy evidence does not support a finding of pneumoconiosis. I further find the x-ray evidence and medical opinion evidence also does not support a finding of pneumoconiosis.

Pneumoconiosis Arising Out Of Coal Mine Employment:

Assuming, **arguendo**, that the claimant established the existence of pneumoconiosis, he must still prove that his pneumoconiosis arose, at least in part, out of his coal mine employment. 20 C.F.R. § 718.203(a). For a miner who suffers from pneumoconiosis and was employed for ten or more years in one or more coal mines, it is presumed that his pneumoconiosis arose out of his coal mine employment. Id. Because Mr. Poole worked for 14 years in coal mine employment, he is entitled to the rebuttable presumption that his pneumoconiosis, if he had it, arose out of coal mine employment.

Total Disability:

Employer's counsel stated at the original hearing that the employer did not contest that Mr. Poole was totally disabled. (1Tr. 21). I note that this concession is bolstered by the four pulmonary function studies of record, all of which produced qualifying values, and the six blood gas studies of record, all of which yielded qualifying results. 20 C.F.R. § 718.204(b)(2)(i) and (ii). Therefore, I find that Mr. Poole was totally disabled prior to his death.

Total Disability Due to Pneumoconiosis:²¹

Unless one of the presumptions at §§ 718.304, 718.305, or 718.306 is applicable, a miner with fewer than fifteen years of coal mine employment, must establish that his or her total disability is due, at least in part, to pneumoconiosis. The Benefits Review Board has held that "[i]t is [the] claimant's burden pursuant to § 718.204 to establish total disability due to pneumoconiosis . . . by a preponderance of the evidence."

²¹ The 2001 amended version of § 718.204 requires that a claimant establish that pneumoconiosis had a "material adverse effect," that is, was a "substantial cause," of his total disability. The District of Columbia Circuit Court concluded that this provision could not be retroactively applied; rather, it applies only to claims filed after January 19, 2001. National Mining Ass'n. et al. v. Dept. of Labor, Case No. 01-5278 (D.C. Cir. June 14, 2002). Therefore, the amended regulation is not applicable here.

Baumgartner v. Director, OWCP, 9 BLR 1-65, 1-66 (1986); Gee v. Moore & Sons, 9 BLR 1-4, 1-6 (1986) (en banc).

The Board requires that pneumoconiosis be a "contributing cause" to the miner's disability. Scott v. Mason Coal Co., 14 BLR 1-37 (1990), overruling Wilburn v. Director, OWCP, 11 BLR 1-135 (1988).

Medical opinion evidence is the only method available for a claimant to prove total disability due to pneumoconiosis. See Tucker v. Director, OWCP, 10 BLR 1-35, 1-41 (1987). Drs. Winkler, Landers, Zacharia, and Gabrawy did not address total disability causation. Dr. Hebert opined that Mr. Poole's "history of pneumoconiosis" was related to his work in coal mines. Dr. Rose opined that pneumoconiosis was a "substantially contributing cause to [Mr. Poole's] total disability . . .," but noted that tobacco smoke was another contributing factor to his obstructive impairment and likely played a more significant role in the lung disease.

Dr. Emory opined that the miner's disability was due to fifty years of smoking. Dr. Fino also linked the miner's total disability solely to smoking. Finally, Dr. Kress referred to emphysema as a "severely disabling" condition in Mr. Poole and attributed its existence to tobacco use. Dr. Kress specifically disagreed with Dr. Rose's report which he felt "seems to mention tobacco smoke as an afterthought, rather than the direct cause of [Mr. Poole's] demise." He opined that the emphysema was "fully explained by [Mr. Poole's] extensive tobacco use."

For reasons previously discussed, I accord greater weight on the opinions of Drs. Emory, Fino, and Kress. Dr. Emory provided a thorough examination of Mr. Poole, and his report is well-documented. Drs. Fino and Kress reviewed the medical evidence of record to date, and both maintain excellent credentials in the field of pulmonary diseases. Accordingly, I find that the evidence fails to establish that pneumoconiosis was a contributing cause to Mr. Poole's disability by a preponderance of the evidence.

Survivor's Claim

Death Due to Pneumoconiosis:

In order to be eligible for benefits, Mrs. Poole must prove that her husband's death was caused by pneumoconiosis. Under §718.205(c), death will be considered to be due to

pneumoconiosis if the medical evidence establishes that pneumoconiosis was the cause of the miner's death or where pneumoconiosis was a substantially contributing cause or factor leading to the miner's death. Pneumoconiosis is a "substantially contributing cause" of a miner's death if it hastens the miner's death. 20 C.F.R. §718.205(c)(5). The Benefits Review Board has held that "death will be considered to be due to pneumoconiosis where the cause of death is significantly related to or significantly aggravated by pneumoconiosis. Foreman v. Peabody Coal Co., 8 BLR 1-371, 1-374 (1985).

Survivors are not eligible for benefits where the miner's death was caused by a traumatic injury, or the principal cause of death was a medical condition not related to pneumoconiosis, unless the evidence establishes that pneumoconiosis was a substantially contributing cause of death. 20 C.F.R. §718.205(c)(4); Neeley v. Director, OWCP, 11 BLR 1-85 (1988). In order to recover benefits, Mrs. Poole must prove that the miner had pneumoconiosis, that the disease arose out of coal mine employment and that pneumoconiosis hastened her husband's death in some manner. 20 C.F.R. § 718.205(a)(1)-(3). Of course, because I have already found that the evidence fails to establish the existence of pneumoconiosis, Mrs. Poole cannot prove that the disease hastened her husband's death. Nevertheless, I will analyze the evidence under the proper standard.

The death certificate provides no assistance in this case. It was not signed by a physician, and the cause of death is listed obliquely as "unspecified natural causes." During deposition, Dr. Gabrawy opined that the main cause of death was coronary artery disease, although he indicated that "bronchopneumonia" impaired the lungs' ability to provide oxygen which "added to the heart impairment together." Dr. Emory found no evidence that the miner's death was a consequence of coal workers' pneumoconiosis. Dr. Fino opined that coal mine dust inhalation did not contribute to, cause, or hasten the miner's death. Likewise, Dr. Kress asserted that there was no evidence to suggest that pneumoconiosis hastened, caused, or contributed to the miner's death. Dr. Winkler opined that anthracosilicosis and cigarette smoking contributed to Mr. Poole's demise; similarly, Drs. Hebert and Rose concluded that pneumoconiosis was a substantially contributing factor leading to the miner's death.

I place greater weight on the opinions of Drs. Gabrawy, Emory, Fino, and Kress because they are consistent with my

finding of no pneumoconiosis, the miner's extensive smoking history, his presenting complaints, and treating physicians' physical findings. Moreover, there is no evidence in support of a finding that Mr. Poole's death was due in any way to pneumoconiosis. Although the opinions of Drs. Winkler, Hebert, and Rose arguably support such a finding, I previously found their opinions are not entitled to persuasive weight. Consequently, I find that Mrs. Poole has failed to establish that pneumoconiosis hastened her husband's death.

Entitlement:

As the claimant-miner has failed to establish the existence of pneumoconiosis or that he was totally disabled by the disease, I find that he is not entitled to benefits under the Act. Furthermore, as the claimant-widow has failed to establish that her husband's death was due to pneumoconiosis, I find that she is not entitled to benefits under the Act.

Attorney's Fees:

The award of an attorney's fee under the Act is permitted only in cases in which the claimant is found to be entitled to the receipt of benefits. Because the benefits are not awarded in this case, the Act prohibits the charging of any attorneys' fee to the claimant for legal services rendered in pursuit of benefits.

ORDER

It is therefore **ORDERED** that the claims of Frances E. Poole and Sterling Poole for benefits under the Act are **DENIED**.

ORDERED this 28TH day of July, 2005, at Metairie, Louisiana.

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LEE J. ROMERO, JR.
Administrative Law Judge

NOTICE OF APPEAL RIGHTS

Pursuant to 20 C.F.R. § 725.481, any party dissatisfied with this Decision and Order may appeal it to the Benefits Review Board within 30 days from the date of this decision, by filing notice of appeal with the Benefits Review Board, P.O. Box 37601, Washington, D.C. 20013-7601. **A copy of a notice of appeal must also be served on Donald S. Shire, Esquire, Associate Solicitor for Black Lung Benefits. His address is Frances Perkins Building, Room N-2117, 200 Constitution Avenue, N.W., Washington, D.C. 20210.**